EVALUATION OF EFFECT OF EPIDURAL ANAESTHESIA ON PULMONARY FUNCTIONS IN PATIENTS UNDERGOING UPPER ABDOMINAL SURGERY UNDER GENERAL ANAESTHESIA

A COMPARATIVE STUDY ON 82 PATIENTS

DISSERTATION SUBMITTED FOR THE DEGREE OF

DOCTOR OF MEDICINE

BRANCH X (ANAESTHESIOLOGY)

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CHENNAI

TAMILNADU

BONAFIDE CERTIFICATE

This is to certify that this dissertation entitled **"EVALUATION OF EFFECT OF EPIDURAL ANAESTHESIA ON PULMONARY FUNCTIONS IN PATIENTS UNDERGOING UPPER ABDOMINAL SURGERY UNDER GENERAL ANAESTHESIA"** is a bonafide record work done by Dr.M.Radhika under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfilment of University regulation for MD, Branch X – Anaesthesiology.

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DECLARATION

I **Dr. M. RADHIKA** solemnly declare that this dissertation titled **"EVALUATION OF EFFECT OF EPIDURAL ANAESTHESIA ON PULMONARY FUNCTIONS IN PATIENTS UNDERGOING UPPER ABDOMINAL SURGERY UNDER GENERAL ANAESTHESIA"** has been done by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree or diploma to any other University or board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of Doctor of Medicine degree Branch –X (Anaesthesiology) to be held in April 2012.

Place: Madurai Dr. M. RADHIKA

Date:

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CONTENTS

INTRODUCTION

It is a well known fact that surgery and anaesthesia perturb the normal physiology of a patient. All the systems in the human body are affected and the pulmonary system is not an exception. Many factors responsible for postoperative pulmonary dysfunction include anaesthetic, surgical and patient factors.

Postoperative pulmonary dysfunction is an entity with multifactorial causes. The patient anaesthesia surgery triad act in an interdependent way in the pathogenesis of this dysfunction.

Anaesthetic factors can be directly related to the disruption of the normal activity of the respiratory muscles, disruption that begins with the induction of anaesthesia and that may continue into the postoperative period. At high doses, anaesthetics attenuate the activities of all respiratory muscles. However, at moderate depths of anaesthesia, anaesthetics may produce respiratory depression by altering the distribution and timing of neural drive to the respiratory muscles, rather than by producing a global depression of activity.

Thus, perioperative respiratory muscle dysfunction in many cases is more a matter of a lack of coordination than a lack of overall activity. As with other complex systems, this lack of

coordination reduces efficiency, producing hypoventilation. In addition, alteration of the chest wall dynamics and dimensions alters the underlying lung, decreasing the functional residual capacity and producing atelectasis in dependent lung regions. These and other effects produce the ventilation-perfusion inequalities characteristic of anaesthesia.

Other indirect factors that may contribute to postoperative pulmonary complications include

1. Reflex stimulation during airway instrumentation and release of inflammatory mediators by drug administration, increasing airway resistance and limiting expiratory gas flow from the lung; if severe this can produce hyperinflation with risk of barotraumas and gas exchange abnormalities.
2. Impairment of normal mucociliary transport by anaesthetic gases and endotracheal intubation which may delay clearance of pathogens and promote retention of secretions.
3. Impairment of lung inflammatory cell's function by prolonged anaesthesia and surgery, which could increase susceptibility to postoperative infections.

4. Impaired upper airway reflexes postoperatively, with may increase the risk of aspiration, and
5. Incomplete reversal of neuromuscular blockade.

These effects of anaesthesia can persist into the postoperative period, and through different mechanisms, the effects of surgical trauma also come into play. These are most pronounced following thoracic and abdominal surgery, and arise from at least three mechanisms.

First, functional disruption of respiratory muscles by incisions, even after surgical repair, may impair their effectiveness.

Second, postoperative pain may cause voluntary limitation of respiratory motion. Finally, stimulation of the viscera, markedly decreases phrenic motor neuron output and changes the activation of other respiratory muscles, in general acting to minimize diaphragmatic descent.

Apart from the above anaesthetic and surgical reasons, patient factors including age, pre existing lung diseases, smoking, effort tolerance and obesity play major roles in determining the postoperative pulmonary dysfunction.

Postoperative pulmonary dysfunction may delay recovery and if severe can be life threatening. Hypoxia may impair wound healing and cognitive function especially in the elderly. Atelectasis predisposes patients to chest infections and chest infection predisposes to respiratory failure.

During recovery from general anaesthesia, there is a gradual increase in neural and muscular activity. The resting pulmonary indices like tidal volume recover in parallel with the reduction in plasma levels of drugs. But the forced indices of pulmonary function like FVC,PEFR,MBC which help in maintaining airway, clearing secretions, prevention of atelectasis and hypoxia recover in an unpredictable fashion that is dependent on patient, anaesthesia and surgery factors.

It takes variable period of time for patients to regain their pre operative levels of pulmonary function. Before the recovery, they succumb to hypoxia, infection, poor wound healing and retained secretions. It is the duty of the anaesthesia care provider to reduce the impact of factors that cause the pulmonary dysfunction.

Hence, it is clear that, when postoperative patients are relatively pain free, their pulmonary function is improved. They can readily expand their chest, breathe deeply, cough well and cooperate with physical

therapy. They are therefore less likely to develop atelectasis, hypoxia and pulmonary infection and more likely to recover quickly and uneventfully.

Pain can be alleviated via intravenous opioids, NSAIDS or neuraxial blockade. Since the former has their own disadvantages like respiratory depression, gastritis and PONV, the beneficial aspects of neuraxial blockade has made its role in improving pulmonary function in the postoperative period overcoming those disadvantages. Of neuraxial analgesic techniques, epidural offers more versatile advantages of less hemodynamic effect, extendable duration, depth and area of analgesia.

They also provide better suppression of surgical stress, positive effect on postoperative nitrogen balance, more stable cardiovascular haemodynamics, reduced blood loss, better peripheral vascular circulation and better postoperative pain relief.

AIM OF THE STUDY

This study was undertaken with the aim of evaluating the effect of epidural anaesthesia on pulmonary functions in patients undergoing upper abdominal surgeries under general anaesthesia. The dynamic parameters of lung function evaluated in the study are,

1. FEV_1 [Forced expiratory volume at 1^{st} second]
2. FVC [Forced vital capacity]
3. PEFR [Peak expiratory flow rate]

PULMONARY FUNCTION TEST

Pulmonary function testing has come into widespread use since the 1970s. This has been facilitated by several developments. From cumbersome spirometers, miniaturization and advances in computer technology, microprocessor devices have made portable and automated spirometers with fewer moving parts. Testing equipment, patient maneuvers, and testing techniques have become widely standardized throughout the world through the efforts of professional societies. Widely accepted normative parameters have been established.

Pulmonary function testing is a valuable tool for evaluating the respiratory system, representing an important adjunct to the patient history, various lung imaging studies, and invasive testing such as bronchoscopy and open-lung biopsy. Insight into underlying pathophysiology can often be gained by comparing the measured values for pulmonary function tests obtained on a patient at any particular point with normative values derived from population studies. The percentage of predicted normal is used to grade the severity of the abnormality. It is necessary for stratifying preoperative risk, and for diagnosing common diseases such as asthma and chronic obstructive pulmonary disease.

Pulmonary function tests is a generic term used to indicate a battery of studies or maneuvers that may be performed using standardized equipment to measure lung function. Pumonary function tests can include simple screening spirometry, formal lung volume measurement, diffusing capacity for carbon monoxide, and arterial blood gases. These studies may collectively be referred to as a complete pulmonary survey. Before a spirogram can be meaningfully interpreted, it is necessary to understand the graphic data (the volume-time curve and the flow-volume loop) to ascertain whether the study meets certain well-defined acceptability and reproducibility standards. Tests that fail to meet these standards can provide useful information about minimum levels of lung function, but, in general, they should be interpreted cautiously. The interpretation strategy usually involves establishing a pattern of abnormality (obstructive, restrictive, or mixed), grading the severity of the abnormality, and assessing trends over time.

PHYSIOLOGY

Basic concepts of normal pulmonary physiology that are involved in pulmonary function testing include mechanics (airflows and lung volumes), the ventilation-perfusion interrelationship, diffusion and gas exchange, and respiratory muscle or bellows strength. Ventilation is the

process of generating the forces necessary to move the appropriate volumes of air from the atmosphere to the alveoli to meet the metabolic needs of the body under a variety of conditions. Simply, the contraction of the diaphragm and other inspiratory muscles expands the thorax, generating negative pressure in the pleural space. One component of pleural pressure, known as transpulmonary pressure causes a flow of air into the airways and lungs (inspiration). When the transpulmonary and alveolar pressures equilibrate, airflow stops, the inspiratory muscles relax, and the lungs and chest wall elastic recoil raise pleural pressure, forcing air out of the lungs (expiration).

With a forced exhalation, the early portion of the spirometry maneuver is characterized by high flows, mostly from large airways, and the latter portion is characterized by low flows with a larger contribution from the smaller airways. Forced inspiration is generally not flow limited and is a function of overall muscular effort. In contrast, a variety of factors affect expiratory flow, including the overall driving pressure, airway diameter, overall distensibility of the lungs and chest wall, dynamic airway collapse (from a flow-limiting segment), and muscular effort.

PULMONARY FUNCTION TEST INDICES

1. BEDSIDE TESTS
2. STATIC TESTS

   Lung voumes and capacities

3. DYNAMIC TESTS

   Forced spirometry

   Maximum breathing capacity

   Flow volume loops

   Assessment of respiratory muscle strength

4. COMPLIANCE TESTS

   Static compliance

   Dynamic compliance

   Chest wall compliance

In this study forced spirometry was used to compare the pulmonary function in two groups. The following three forced indices were used:

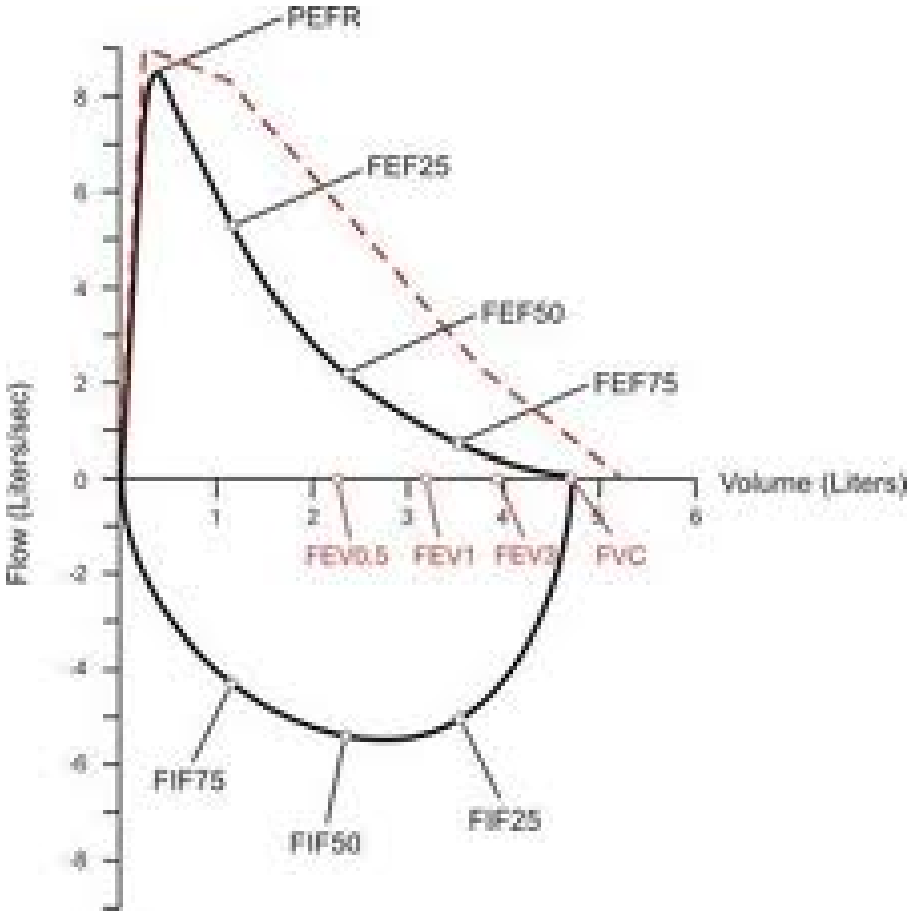
1. FEV_1
2. FVC
3. PEFR

FORCED EXPIRATORY VOLUME IN 1 SECOND

They correlate with airway diameter and expiratory strength. The FEV_1 is the most widely used parameter to measure the mechanical properties of the lungs. In normal persons, the FEV_1 accounts for the greatest part of the exhaled volume from a spirometric maneuver and reflect mechanical properties of the large and the medium-sized airways. In a normal flow-volume loop, the FEV_1 occurs at about 75% to 85% of the FVC. This parameter is reduced in obstructive and restrictive disorders.

FORCED VITAL CAPACITY

FVC is a measure of lung volume and expiratory strength together. It is usually reduced in diseases that cause the lungs to be smaller. Such processes are generally termed restrictive and can include disorders of the lung parenchyma, such as pulmonary fibrosis, or of the bellows, including kyphoscoliosis, neuromuscular disease, and pleural effusion. However, a reduction in FVC is not always due to reduced total volumes and can occur in the setting of large lungs hyperinflated due to severe airflow obstruction and air trapping, as in emphysema. Reduced FVC can occur despite a normal or increased total lung volume. Therefore, FVC is not a reliable


PEFR
FEF25
FEF50
FEF75
Flow (Liters/sec)
Volume (Liters)
8
6
4
2
0
-2
-4
-6
-8
1
2
3
4
5
6
FEV0.5
FEV1
FEV3
FVC
FIF75
FIF50
FIF25

indicator of total lung capacity or restriction, especially in the setting of airflow obstruction.

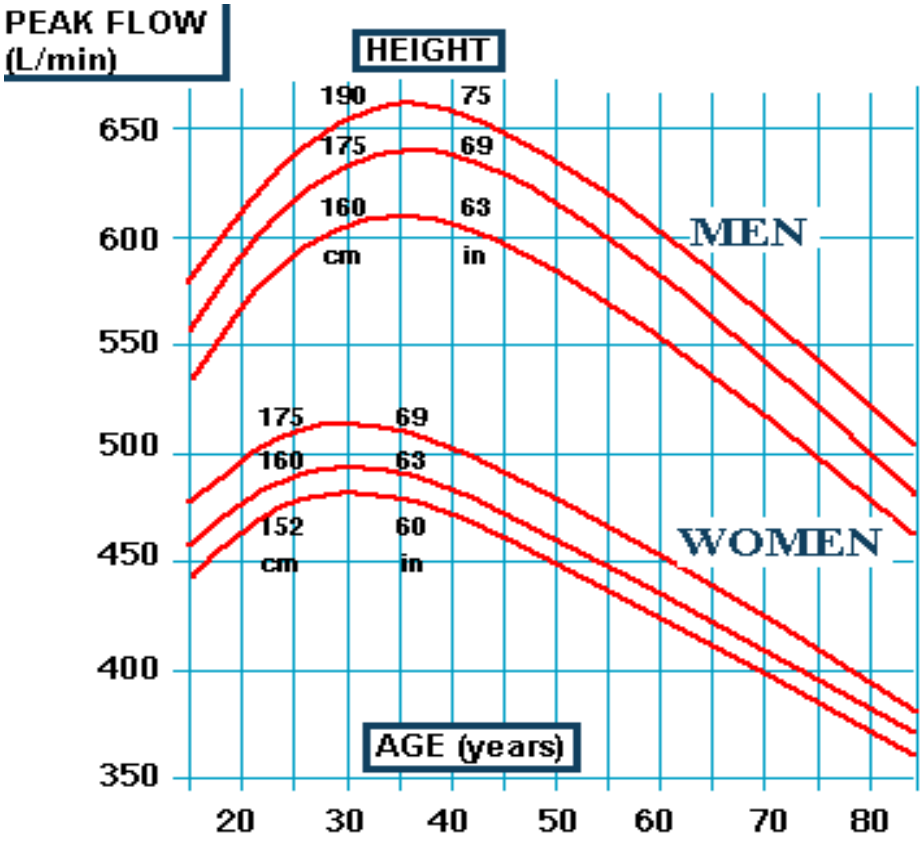
The overall accuracy of the FVC for restriction is about 60%. FEV_1/FVC Ratio, also called Tiffeneau index is used in the diagnosis of obstructive and restricted lung disease. It represent the proportion of forced vital capacity exhaled in first second. Normal value is approximately 80%.

PEAK EXPIRATORY FLOW RATE

Peak expiratory flow rate (PEFR) is the maximum flow rate generated during a forceful exhalation, starting from full lung inflation. Peak flow rate primarily reflects large airway flow and depends on the voluntary effort and muscular strength of the patient. Normal value is 450 – 700L/min.

Maximal airflow occurs during the effort-dependent portion of the expiratory maneuver, so low values may be caused by a less than maximal effort rather than by airway.

PEF is appreciably larger if the maneuver is performed without pause immediately after the inspiration than if it is performed after a pause. If the maneuver is performed sufficiently forcefully in most


PEAK FLOW
(L/min)
HEIGHT
190
75
175
69
160
63
cm
in
MEN
175
69
160
63
152
60
cm
in
WOMEN
650
600
550
500
450
400
350
AGE (years)
20
30
40
50
60
70
80

subjects PEF is determined by flow limitation in central, possibly also in more peripheral airways.

Report the largest value of 3 correctly performed maneuvers, but the difference between the largest two should be less than 40 L/min; if the difference is larger, then have up to 2 extra efforts performed. If even then the two largest values differ by > 40 L/min, then report the largest one with a note to the effect that reproducible measurements could not be obtained.

Daily variability in PEF is larger in asthmatics than in healthy subjects Predicted values of PEF are of little use in detecting lung disease, as variability between healthy subjects is large, so that a value within the normal range certainly does not rule out the presence of airway obstruction.

In severe obstructive lung disease rapid and extensive dynamic airway compression during the forced maneuver contribute appreciably to initial maximum expiratory flow from the lungs; the severity of airway obstruction then tends to be underestimated from PEF.

PULMONARY FUNCTION TESTING MEASUREMENT

Accurate spirometric pulmonary function testing has three components:

1. A frequently calibrated spirometer that accurately records exhaled volume in relation to time during the forced expiration.
2. A subject who inspires maximally and exhales as forcefully, smoothly, and completely as he can
3. A technician who can calibrate the spirometer , coach the subject to perform maximal efforts and detect poor subject effort or expirations that are not continuous, maximal and completely recorded.

PERFORMANCE OF THE TEST

1. Explain the test
2. Prepare the subject
3. Enquire about smoking, recent illness, medication use, etc.
4. Measure weight and height without shoes
5. Wash hands
6. Instruct and demonstrate the test to the subject
7. In this study, test was performed in the supine posture
8. Have subject assume the correct posture
9. Attach nose clip, place mouthpiece in mouth and close lips around the mouthpiece.

10. Inhale completely and rapidly with a pause of ,1 s at TLC
11. Exhale maximally until no more air can be expelled.
12. Repeat instructions as necessary, coaching vigorously
13. Repeat for a minimum of three manoeuvres; no more than eight are usually required.

SPIROMETER

The spirometer used in this study for testing pulmonary function was

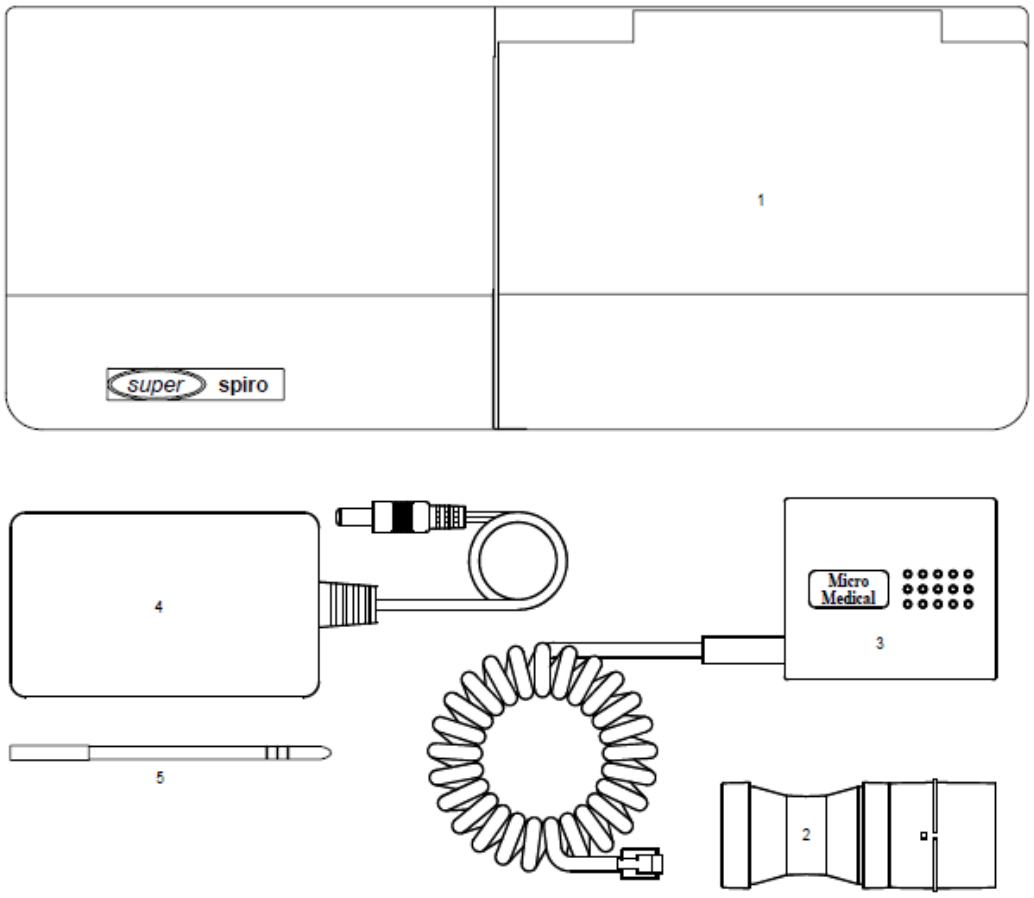
MICRO MEDICAL SUPER SPIRO

The Micro Medical SuperSpiro is a powerful and sophisticated desktop spirometer. This comprehensive suite of analysis and management software is packaged in a sleek, compact desktop unit with a built-in colour monitor and thermal printer powered by a rechargeable internal battery pack. Extra features include USB support and touch-screen facilities and a modern graphical user-interface.

The SuperSpiro includes a patient database which can store up to 3000 patients. The spirometer uses the Micro Medical Digital Volume Transducer, an extremely stable form of volume transducer, which measures expired air directly at B.T.P.S (Body Temperature and Pressure with Saturated water vapour)[24] thus avoiding the inaccuracies of temperature corrections. This transducer is insensitive to the effects of condensation and temperature and avoids the need for individual calibration prior to performing a test.

The SuperSpiro has many advanced features including a colour LCD display giving real time Flow/Volume or Volume/Time curves, user

MICRO MEDICAL SUPER SPIRO



1 Super Spiro

2 Micro Medical digital volume transducer

3 Transducer housing

4 Universal AC adapter

5 Stylus x 3

6 USB cables

7 RS-232 cable

customisation of instrument functions, predicted values, and the ability to carry out pre and post bronchodilator, and bronchial challenge testing.

The spirometry measurements, Flow/Volume loop, and Volume/Time curve may be printed at the time of testing or at any time subsequently from memory. One unique feature is the ability to expand the capabilities of the instrument by simply downloading new software modules supplied with other transducers.

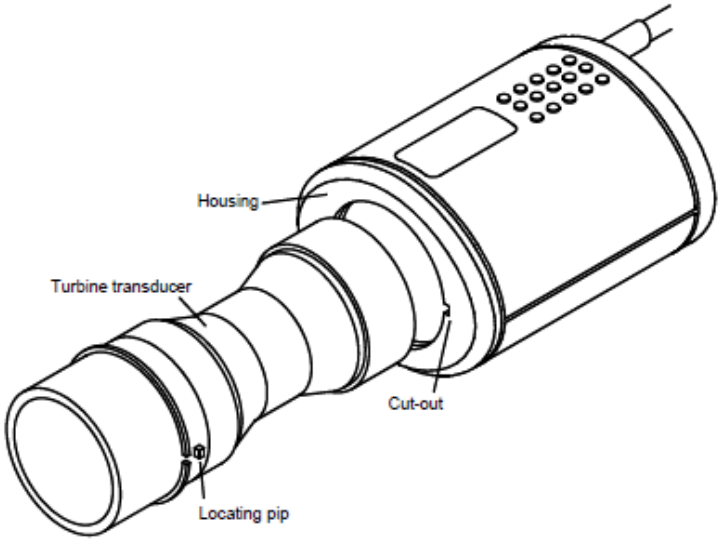
It is recommended that only the required tests be activated to reduce the selections required during operation. The spirometry test may be performed with force or relaxed expiration and inspiration, with or without prior tidal breathing.

For spirometry tests - the age range should be from 7 to 110 years, and height from 110cm to 250cm.

Recording of forced indices

The spirometry screen is displayed showing the predicted Flow/ Volume curve as a dashed line, or a shaded area, depending on the setting. The predicted curve will only be displayed if the patient details have been entered. The arrows in the top right hand corner of the screen indicate that the left and right cursor keys are active. In this case they are used to change the display between the full or expiratory only flow/volume loop,

TRANSDUCER



the full or expiratory only volume/time curve, or the child incentive display. A spirometry test may be performed with or without tidal breathing through the transducer.

Instruct the patient to breathe in until their lungs are completely full, then seal their lips around the mouthpiece and blow out as hard and as fast as possible until they cannot push any more air out. Then to breathe in fully, immediately after the expiratory manoeuvre, thus completing the flow volume loop.

The Flow/Volume loop is displayed as the patient performs the manoeuvre. At the end of the test, values for FEV_1, FVC and PEF are displayed, together with the Flow/Volume loop, and a manoeuvre quality check to allow a decision to be made to accept or reject this blow.

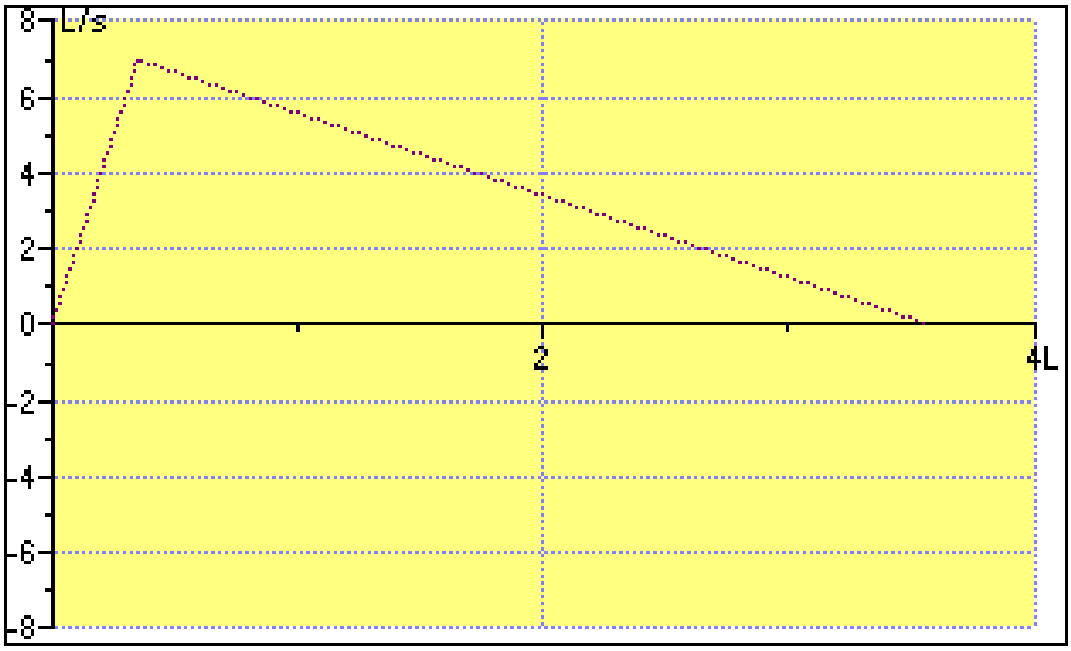
Quality Checks

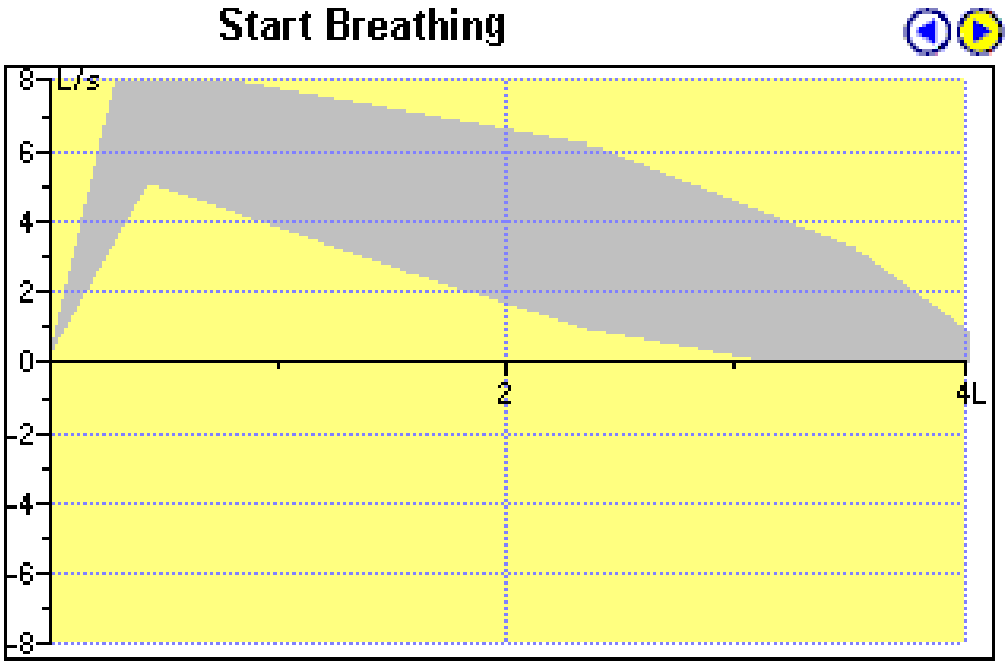
There are five quality checks[24] performed on each spirometry manoeuvre to determine its acceptability. They are as follows:

If the patient performs an acceptable manoeuvre 'Good blow' is displayed at the top of the screen.

If the back extrapolated volume (BEV) was greater than 150ml then 'Slow start' will appear. This indicates that the patient did not blast out the air quickly and evenly during the forced expiration.

START BREATHING

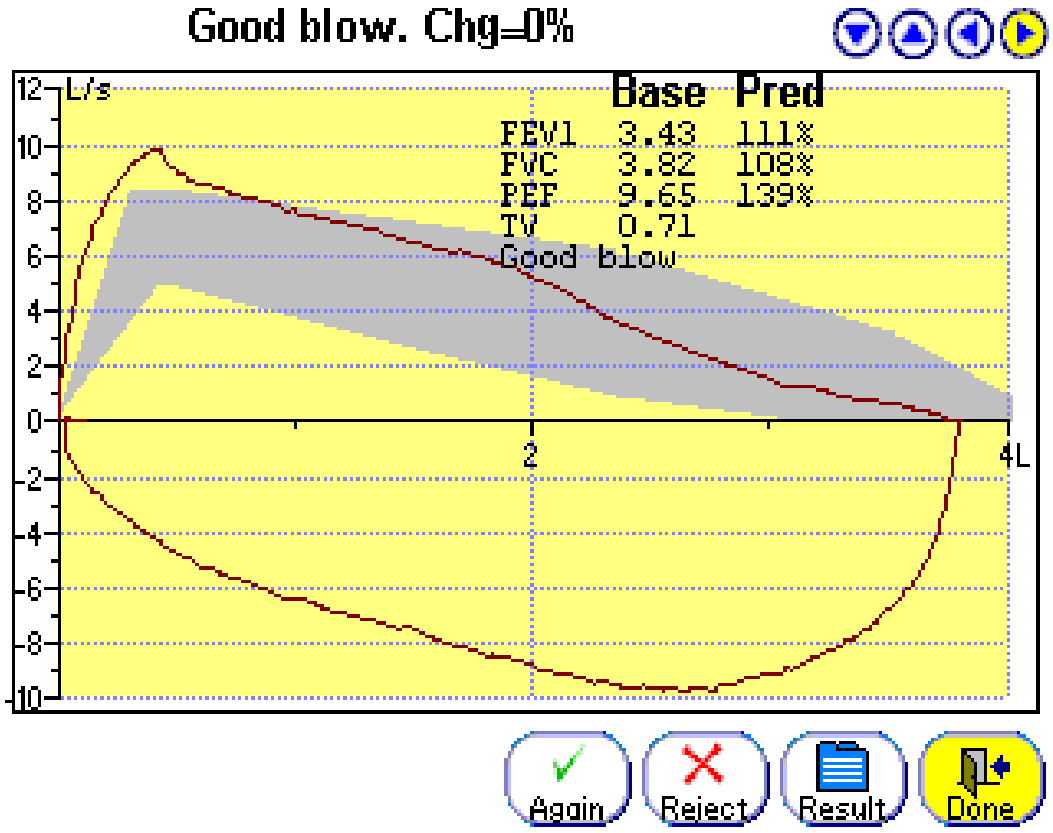




If the time to peak flow (PEFT) was greater than 120msec then ‘poor effort’ will appear indicating a sluggish effort during the forced expiration.

If the forced expiratory time (FET) was less than 6 seconds and the change in exhaled volume during the last half second was more than 100mL Abrupt end’ will appear. The patient had stopped exhaling prematurely.

If the expiratory flow exhibited a secondary peak then ‘Cough detected’ will be displayed.


Good blow. Chg=0%
L/s
Base Pred
FEV1 3.43 111%
FVC 3.82 108%
PEF 9.65 139%
TV 0.71
Good blow
12
10
8
6
4
2
0
-2
-4
-6
-8
-10
2
4L
Again
Reject
Result
Done

GENERAL ANAESTHESIA AND RESPIRATORY OUTCOME

Anesthesia causes an impairment in pulmonary function, whether the patient is breathing spontaneously or is ventilated mechanically after muscle paralysis. Impaired oxygenation of blood occurs in most patients who are anesthetized. Lung function remains impaired postoperatively, and clinically significant pulmonary complications can be seen in 1% to 2% after minor surgery in up to 20% after upper abdominal and thoracic surgery.

In 1964, Nunn showed that during routine anaesthesia and spontaneous ventilation, gas exchange was altered by shunt and uneven ventilation perfusion ratios.

In the 1980s, atelectasis was shown by computed tomography in anaesthetized patients—neonates as well as adult.[20] Lung densities were seen in anaesthetized children and were called 'confluent high absorptive areas' but such areas were not found in the scans performed under sedation.

Thus clearly general anaesthesia poses the following various pulmonary complications:

1. Atelectasis
2. Hypoxemia

3. Ventilation perfusion mismatch
4. Respiratory drive reduction
5. Reduced compliance and increased resistance.

1.ATELECTASIS

These occur in the most dependent parts of the lungs and are found in almost 90% of all patients who are anaesthetized. They develop with both intravenous and inhalational anaesthesia and whether the patient is breathing spontaneously or is paralysed and ventilated mechanically.

Good correlations have been found between gas exchange impairment and the amount of atelectasis. Most atelectasis occurs near the diaphragm in the supine patient and less towards the apex. Atelectasis may contain 15–20% of the lung tissue close to the diaphragm and about 10% of the total lung tissue. Atelectasis can persist for two days after major surgery.

Pulmonary atelectasis may be caused by a variety of factors, which have been classified into three basic mechanisms. Compression atelectasis occurs when the transmural pressure distending the alveolus is reduced. Absorption atelectasis occurs when less gas enters the alveolus than is removed by uptake by the blood. Loss-of-surfactant atelectasis occurs when the surface tension of an alveolus increases because of reduced

surfactant action. Any of these factors may contribute to atelectasis during anaesthesia and the postoperative period.

Compression Atelectasis

The rapid formation of atelectasis on induction of anaesthesia, and the fast reappearance after discontinuation of PEEP suggested that the atelectasis was caused by compression of lung tissue rather than by resorption of gas behind occluded airways. Loss of inspiratory muscle tone is an important factor in atelectasis formation. It may thus be that the greater abdominal pressure is more easily transmitted into the thoracic cavity when the diaphragm has a reduced tone or is paralysed, as during anaesthesia.[20]

The classic study by Froese and Bryan showed that diaphragm motion in spontaneously breathing normal volunteers is changed when the volunteers are paralysed with neuromuscular blocking agents. Hence Froese and Bryan concluded that in the supine position during spontaneous ventilation, the dependent part of the diaphragm had the greatest displacement. However, after neuromuscular block and positive pressure ventilation, exactly the opposite was seen: the non-dependent part had the greatest displacement.

Also, Krayer et al using CT scans, found altered diaphragmatic motion during general anaesthesia and mechanical ventilation. In addition, Warner et al found alterations in the end-expiratory position of chest wall structures during general anaesthesia and Reber et al showed that general anaesthesia induced a cephalad displacement of the most dorsal part of the diaphragm. Thus, compression atelectasis occurs during general anaesthesia and is caused by chest geometry and diaphragm position and motion.

Absorption atelectasis

Absorption atelectasis can occur by two mechanisms. The first mechanism is complete airway occlusion, which creates a pocket of trapped gas in the distal lung unit.[20] The pressure in the pocket initially is close to atmospheric pressure. Mixed venous blood continues to perfuse the pocket, and since the sum of the gas partial pressures in the mixed venous blood is subatmospheric, gas uptake from the pocket by the blood continues and the pocket collapses.

The second mechanism is when the inspired V/Q ratio is less than a critical value. If the inspired V/Q ratio of a lung unit is reduced, a point is reached where the rate at which inspired gas entering the alveolus is exactly balanced by gas uptake from the alveolus into the blood.[20] This point is

known as the critical V/Q ratio. If the inspired V/Q ratio is less than this, the lung unit will collapse. This is likely when Fi_{O2} is high and the gas uptake is large.

Loss-of-surfactant atelectasis

Atelectasis, can impede surfactant function so that a region is prone to collapse again after having been reopened.[20] A vital capacity manoeuvre may promote surfactant production or release, and distribution of surfactant over the alveolar surface may cause a longer lasting protection against new collapse. Indeed, it has been shown that large gasps increase the proportion of active forms of alveolar surfactant.

2.HYPOXEMIA

Several factors contribute to hypoxemia in general anaesthesia.

1. After induction of general anesthesia, there is a loss of the inspiratory tone and an appearance of end-expiratory tone in the abdominal expiratory muscles at the end of exhalation. The end-expiratory tone in the abdominal expiratory muscles increases intra-abdominal pressure, forces the diaphragm cephalad, and decreases FRC. Thus, after the induction of general anesthesia, there is loss of a force tending to maintain lung volume and gain of a force tending to decrease lung volume.

2. In the upright position, the FRC and the position of the diaphragm are determined by the balance between the lung elastic recoil pulling the diaphragm cephalad and the weight of the abdominal contents pulling it caudad. There is no transdiaphragmatic pressure gradient. But in supine position, the diaphragm separates two compartments of markedly different hydrostatic gradients. On the thoracic side, pressure increases by approximately 0.25 cm H2O/cm of lung height, and on the abdominal side by 1.0 cm H2O/cm of abdominal height. Hence in supine position, progressively higher transdiaphragmatic pressures must be generated toward dependent parts of the diaphragm to keep the abdominal contents out of the thorax. In the unparalyzed patient, this tension is developed either by passive stretch and shape changes of the diaphragm (causing an increased contractile force) or by neurally mediated active tension. With acute muscle paralysis, neither of these two mechanisms can operate, and a shift of the diaphragm to a more cephalad position occurs . The latter position must express the true balance of forces on the diaphragm, unmodified by any passive or active muscle activity. The cephalad shift in the FRC position of the diaphragm owing to expiratory muscle tone during general anesthesia is equal to the shift observed during paralysis. The equal shift suggests that the pressure on the diaphragm caused by an

increase in expiratory muscle tone during general anesthesia is equal to the pressure on the diaphragm caused by the weight of the abdominal contents during paralysis.

3. The overall reduction in all components of lung volume during anesthesia results in a reduced caliber of airway, which increases airway resistance and any tendency toward airway collapse. The decreases in FRC caused by the supine position and the induction of anesthesia lead to increased resistance of the airway seen in the healthy anesthetized patient.
4. In the supine position, the abdominal contents force the diaphragm cephalad and reduce the FRC. The Trendelenburg position allows the abdominal contents to push the diaphragm further cephalad, so that the diaphragm not only must ventilate the lungs, but it also must lift the abdominal contents out of the thorax. The result is a predisposition to decreased FRC and atelectasis. Increased pulmonary blood volume and gravitational force on the mediastinal structures are additional factors that may decrease pulmonary compliance and FRC. In the steep Trendelenburg position, most of the lung may be below the left atrium and therefore in a zone 3 or 4 condition. As such, the lung may be susceptible to the development of pulmonary interstitial edema.

5. Acute arterial hypoxemia from a transient right-to-left shunt through a patent foramen ovale has been described, particularly during emergence from anesthesia. However, unless there is a real-time technique of imaging the cardiac chambers (color-flow Doppler mapping), it is difficult to document an acute and transient right-to-left intracardiac shunt as a cause of arterial hypoxemia.

3.VENTILATION-PERFUSION(V/Q)MISMATCH

Ideally, ventilation should match perfusion perfectly (V/Q = 1) at the alveolus, but there is always some mismatch. Both CO_2 elimination and oxygenation of blood are impaired in most patients during anesthesia. The impeded CO_2 elimination can be attributed to increased dead space ventilation. Single-breath washout recordings have demonstrated that "anatomic" dead space is unchanged, indicating that the "alveolar" or physiological dead space must have increased during anesthesia.. General anesthesia tends to increase mismatch in 2 possible ways:

1) High V/Q (> 1 to ∞) occurs when perfusion is low compared with ventilation because of increased dead space. Dead space in an anesthetized patient includes the following:

- Mechanical dead space, or portions of the breathing circuit in which there is no separation of inspired and expired gas streams (excessively long endotracheal tubes or extensions of the Y-connector)
- Anatomic dead space, or airways are not involved in gas exchange.
- Physiologic dead space are those that are being ventilated but not perfused due to low cardiac output, pulmonary arterial hypotension, cardiopulmonary arrest, or pulmonary thromboembolism; increased physiologic dead space can also be classified as a type of perfusional hypoxia

2) Low V/Q (< 1 to 0) occurs when ventilation is low compared with perfusion, and effectively causes a right-to-left shunt. Shunt occurs whenever blood passes from the right to left side of the circulation without exposure to oxygen. Normal shunt fraction is about 5% because venous blood from the myocardial and bronchial circulations empties into the left ventricle.

Recumbency, general anesthesia, and positive intrathoracic pressure predispose to the development of atelectasis, which effectively causes shunting of blood from the right to the left side of the circulation without exposure to oxygen (these alveoli are perfused but not well ventilated).

Oxygen uptake is greatly compromised by shunt, resulting in low PaO2, even with supplemental oxygen. Carbon dioxide elimination is not compromised until the shunt is very severe (> 50%) because of its high diffusibility.

4. REDUCTION OF RESPIRATORY DRIVE

Anesthesia reduces the response to hypoxia. Attenuation of the hypoxic response may be attributed to an effect on the carotid body chemoreceptors. The effect of an anesthesia on respiratory muscles is nonuniform. Rib cage excursions diminish with deepening anesthesia. The normal ventilatory response to CO_2 is produced by the intercostal muscles. Thus, the reduced ventilatory response to CO_2 during anesthesia is due to impeded function of the intercostal muscles.[1]

5. REDUCED COMPLIANCE AND INCREASED RESISTANCE

Total thoracic compliance = 100ml/cm H_2O:

Lung compliance = 200ml/cm H_2o

Thoracic compliance = 200ml/cm H_2o

Static compliance of the total respiratory system (lungs and chest wall) is reduced on average from 95 to 60 ml/cm H_2O during anesthesia. Several studies on lung compliance have been carried out during anesthesia, and the vast majority of studies indicate a decrease in

comparison to the awake state (e.g., static lung compliance fell from a mean of 187 ml/cm H_2O awake to 149 ml/cm H_2O during anesthesia.[1]

There are also studies on resistance of the total respiratory system and the lungs during anesthesia, most of them showing a considerable increase during both spontaneous breathing and mechanical ventilation. The increased lung resistance merely reflects reduced FRC during anesthesia.

EPIDURAL ANALGESIA AND RESPIRATORY OUTCOME

Postoperative pulmonary dysfunction occurs as a result of surgery and anesthesia-related physiologic perturbations and remains a major cause of postoperative morbidity. Thus, techniques that reduce postoperative pulmonary dysfunction may result in improved clinical outcome.

It has recently been suggested that major complications associated with epidural analgesia might be more common than once thought, hence it is of the utmost importance to have a clear view of the real benefits associated with its use. An updated reappraisal of the benefits associated with epidural analgesia could help the clinician to decide when the benefits of an epidural catheter insertion outweigh its inherent risks in a specific patient. The aim of this review is to sum up the benefits of adding epidural analgesia for patients operated on under general anesthesia emphasizing the improvement of postoperative pulmonary function.

Epidural local anesthetics, with or without opioids, provide better postoperative pain relief than systemic opioids. Epidural local anesthetics reduce central sympathetic stimulation, with subsequent

favorable effects on coagulation and homeostasis and on gastrointestinal, metabolic, and immune function.

Epidural analgesia significantly increased forced vital capacity at 24 hours, forced expiratory volume in 1 second at 24 hours, and peak expiratory flow rate at 24 hours . Epidural analgesia significantly increased arterial oxygen pressure at 24 and 72 hours. [14]

Upper abdominal and thoracic incisions significantly reduce post operative pulmonary function. Pulmonary dysfunction after upper abdominal surgery occurs because of pain, abnormal diaphragmatic function, and increased abdominal and lower intercostal muscle tone during exhalation. Pulmonary dysfunction begins with incision and remains undiminished for 7-14 days postoperatively. The most important alteration of respiratory function is decreased functional residual capacity, which begins about 16 h postoperatively, reaches a nadir at 24-48 h, and usually resolves within 1 week. Decreased functional residual capacity may result in atelectasis and ventilation-perfusion abnormalities leading to hypoxemia, pneumonia, and postoperative pulmonary complications. Patients especially at risk for reduction of functional residual capacity and resultant pulmonary complications include those with preexisting pulmonary

disease, upper abdominal and thoracic incisions, advanced age, obesity, and those in severe pain.

Choice of anesthetic technique affects the degree of postoperative pulmonary dysfunction. Use of general anesthesia may briefly exacerbate surgery-induced pulmonary dysfunction. Mechanical ventilation, paralysis, inhaled anesthetics, and opioids all contribute to reduce pulmonary function. In contrast to the exacerbation of postoperative pulmonary dysfunction seen with general anesthesia, TEA has minimal effect on pulmonary function and may offset detrimental changes in pulmonary function induced by general anesthesia

Many studies suggest that postoperative use of epidural analgesia has the potential to reduce pulmonary morbidity by providing better analgesia, improved diaphragmatic function, and reduced frequency and severity of postoperative hypoxemia. Epidural analgesia using local anaesthetic or opioid, and especially both provides better analgesia than systemic opioid, including delivery by intravenous PCA. Because relief of pain improves pulmonary function after abdominal or thoracic surgery, it is not surprising that use of epidural analgesia is associated with improved pulmonary function

when compared with intramuscular and intravenous opioid.[5] But not all studies demonstrating superior analgesia in the epidural group also demonstrate improvements in pulmonary function suggests that factors other than pain are also important etiologic factors for postoperative pulmonary impairment.

Diaphragmatic function is impaired after abdominal or thoracic surgery and may also contribute to pulmonary dysfunction. Diaphragmatic dysfunction appears to result from reflex inhibition of phrenic nerve activity and is not significantly altered by relief of pain. Thus, analgesia provided with parenteral or epidural administration of opioids alone does not produce appreciable improvement in diaphragmatic function. On the other hand, thoracic epidural blockade with local anesthetic may improve postoperative diaphragmatic function.[14] This improvement in function probably results from neural blockade of the inhibitory reflex[22] and perhaps through changes in chest wall compliance. Thus, epidural administration of local anesthetics for postoperative analgesia can result in improved pulmonary function in part through relief of pain but also by improving postoperative diaphragm function.[14]

Pulmonary dysfunction may also produce morbidity through hypoxemia. Episodic hypoxemia is common in the postoperative period, particularly during sleep.[23] Postoperative hypoxemia serves as a marker for patients with postoperative pulmonary morbidity and is associated with myocardial ischemia. Analgesia with either parenteral or epidural opioid is associated with equally high incidences of episodic postoperative hypoxemia. However, use of epidural analgesia with local anesthetics is associated with a reduced incidence and severity of hypoxemia in the early postoperative period perhaps because of improved pulmonary function or reduced sedation.

Postoperative pulmonary dysfunction can be attenuated by the intraoperative use of epidural local anaesthetics. More important, continuation of epidural analgesia with local anaesthetic into the postoperative period may maintain improvements in postoperative pulmonary function.[14] These effects may be attributable to the benefits in addition to analgesia provided by epidural administration of local anaesthetics, such as limitation of the degree of postoperative diaphragmatic dysfunction, improvement of abdominal or chest wall compliance, and limitation of episodes of postoperative hypoxemia.

In summary, the improvements resulting from epidural analgesia can be attributed to:

- Effective pain relief allowing the patient to take deep breaths, cough and cooperate with physiotherapy;
- Blocking of reflexes inhibiting diaphragmatic function, demonstrable after abdominal and thoracic surgery, is likely to have a beneficial effect on pulmonary mechanics;
- Avoidance of high-dose systemic opioids should reduce respiratory depression; and
- Reduction of the stress response to surgery reduces the level of postoperative immunosuppression, which may contribute to a reduction in pulmonary infection.[14]

REVIEW OF LITERATURE

1. **H. Hendolin et al, Acta Anaesthesiologica Scandinavica, Volume 31, Issue 7, pages 645–651, October 1987;**

   A prospective randomized trial was conducted to assess the effect of thoracic epidural analgesia (TEA) on postoperative respiratory function and pulmonary complications in patients undergoing cholecystectomy. One hundred patients were allocated to TEA (n = 30), TEA + general anaesthesia (TEA + GA) (n = 30), or general anaesthesia (GA) (n = 40) groups. Respiratory function was analysed by measuring forced vital capacity (FVC), forced expiratory volume in 1 s (FEV_1), functional residual capacity (FRC), total lung capacity (TLC), peak expiratory flow (PEF) in the supine and sitting postures, and arterial blood gases.They concluded that **TEA significantly prevented the postoperative deterioration of respiratory function as compared with patients who underwent general anaesthesia**. FVC, FEV_1 and PEF decreased by 20% in patients receiving TEA, in contrast to 55% in patients after GA on the day of operation.

2. **Guay Joahne et al, Journal of Anesth. 2006;20(4):335-40**

   They conducted a meta analysis study regarding the benefits of adding epidural analgesia to general anaesthesia in abdominal surgeries.. This study included 5402 patients, of which 2660 had had epidural analgesia and the remaining general anaesthesia alone.They concluded that epidural analgesia reduces the first 24-h morphine consumption, (P = 0.003), **and improves the forced vital capacity (FVC), (P = 0.001) at 24 h**. Also reduces the incidence of arrhythmia, (P = 0.001); (P = 0.002); intensive care unit stay,(P = 0.03); visual analogical pain (VAS) scores at rest, (P < 0.00001) and during movement, (P < 0.00001)

3. **Paul K. Tenenbein et al, Canadian journal of Anaesthesia ;2009, volume 55, no:6, 344-350**

   They conducted a prospective, randomized, controlled trial in fifty patients, undergoing CABG surgery. Twenty-five patients were enrolled in two groups. Patients in the epidural group had significantly less pain on the operative day, and for the subsequent two days. Compared to baseline, the forced expiratory volume in one second was significantly higher in the epidural group, on the first and second postoperative days (43.7±12.2%vs 36.4±12.0%,p<0.002, and 43.3±12.5% vs 38.4±11.0%, p<0.05). There was significantly more atelectasis in the control group, four

hours postoperatively ($p<0.04$); Hence they concluded **that thoracic epidural analgesia decreases postoperative pain and atelectasis and improves pulmonary function** in patients undergoing CABG surgery.

4. **Rigg et al, The Lancet, volume 359, issue 9314, 2002** pg no: 1276 – 82

   A randomized trial was conducted to evaluate epidural anaesthesia and analgesia and its outcome in major abdominal surgery. The study included 915 patients who underwent major abdominal surgeries; they were divided in to two groups – one control group (GA only) and the other epidural group (GA +epidural). They concluded that improvement in analgesia**, reduction in respiratory failure,** and the low risk of serious adverse consequences suggest that many high-risk patients undergoing major intraabdominal surgery will receive substantial benefit from combined general and epidural anaesthesia intraoperatively with continuing postoperative epidural analgesia.

5. **Daniel M Popping et al - Arch Surg. 2008;143(10):990-999;**

   A meta analysis study was done evaluating protective effects of epidural analgesia on pulmonary complications after abdominal and thoracic Surgery. The study was conducted between 1971-2006,in a large number of patients and concluded that the incidence of pneumonia with epidural analgesia remained about 8% but decreased from 34% to 12% with

systemic analgesia (P< .001) ; Also epidural analgesia reduced the need for prolonged ventilation or reintubation, **improved lung function** and blood oxygenation.

6. **Ballantyne et al - Anaesthesia and analgesia March 1998 vol. 86no. 3 598-612**

They conducted a cumulative meta-analysis of randomized, controlled trials on the comparative effects of postoperative analgesic therapies on pulmonary outcome.

They assessed the effects of seven analgesic therapies on postoperative pulmonary function after a variety of procedures: epidural opioid, epidural local anesthetic, epidural opioid with local anesthetic, thoracic versus lumbar epidural opioid, intercostal nerve block, wound infiltration with local anesthetic, and intrapleural local anesthetic. Measures of forced expiratory volume in 1 s (FEV_1), forced vital capacity (FVC), vital capacity (VC), peak expiratory flow rate (PEFR), P_aO_2, incidence of atelectasis, pulmonary infection, and pulmonary complications overall were analyzed. Compared with systemic opioids, epidural opioids decreased the incidence of atelectasis and had a weak tendency to reduce the incidence of pulmonary infections and pulmonary complications overall. Epidural local anesthetics increased

p_aO_2 and decreased the incidence of pulmonary infections and pulmonary complications overall compared with systemic opioids**.**

7. **Park WY, Thompson JS, Lee KK ; Ann Surg. 2001 Oct;234(4):560-9; 569-71.**

   They did a randomized, controlled study on effect of epidural anesthesia and analgesia on perioperative outcome.

   They studied in 1,021 patients who required anesthesia for one of the intraabdominal aortic, gastric, biliary, or colon operations. They were assigned randomly to receive either general anesthesia and postoperative analgesia with parenteral opioids (group 1) or epidural plus light general anesthesia and postoperative epidural morphine (group 2).. Overall, epidural analgesia provides better postoperative pain relief**. Epidural anesthesia and epidural analgesia improve the overall outcome** and shorten the intensive care stay in patients undergoing abdominal aortic operations.

8. **THE MASTER TRIAL**

   Multicentre Australian study of epidural anaesthesia.

   They investigated the influence of perioperative epidural analgesia on postoperative outcome in 888 patients undergoing major abdominal surgery in comparison with a control group who received intravenous

opioid analgesia. They found **decrease in postoperative respiratory failure in the epidural group**. Also mean Visual Analogue Score with coughing was 30 times less than in control group in first 24 hours.

9. **Miller L, Gertel M.American Journal of Surgery. 1976 Mar;131 (3): 291-4.**

They compared the effect of narcotic and epidural analgesia on postoperative respiratory function.

A prospective, randomized comparison was made of the value of meperidine (intravenous) versus epidural analgesia when used for the relief of pain after cholecystectomy in twenty patients without cardiopulmonary disease. Respiratory function was assessed the day before surgery and at 3 to 4 hours and 24 hours after operation by the bedside measurement of expiratory peak flow, vital capacity, and arterial blood gases. The two groups of patients were comparable as to age, height, weight, smoking habits, preoperative peak flow, vital capacity, and duration of operation.

The arterial oxygen tension and oxygen saturation were significantly greater and carbon dioxide tension lower in the epidural analgesia group 24 hours after operation. At this time peak flow rates and vital capacity were not different. However, at 3 to 4 hours postoperatively, **vital**

capacity was significantly greater in the epidural anesthesia group. These findings suggest that epidural analgesia is valuable in the early postoperative period after upper abdominal surgery

10. **Gross et al, Anesthesiology Clinical North America. 2000 Jun;18(2):407-28,** They studied on the role of epidural anesthesia and analgesia in postoperative outcome. They confirmed the benefits of epidural anaesthesia and analgesia in decreasing the incidence of cardiac complications in high-risk patients**;** decreasing incidence of pulmonary complications, specifically pneumonia, atelectasis, and hypoxemia in patients at risk for pulmonary complications; lower incidence of vascular graft occlusion after lower extremity revascularization; lower incidence of DVT and pulmonary embolus; suppression of the neuroendocrine stress response; and earlier return of gastrointestinal function**.**

11. **Groeben et al, Journal of Anaesthesia ; Volume 20, Number 4, 290- 299, 2006**

They concluded in their article -epidural anesthesia and pulmonary function as follow:

It has been shown that patients who undergo major surgery, such as cardiac surgery, under combined **anesthesia with epidural anesthesia and epidural postoperative analgesia are able to perform VC and**

FEV1 measurements within 1 hour after extubation, while patients without epidural anesthesia and analgesia, and treated with intravenous administration of opioids only, were not able to perform pulmonary function tests in the first hour. This effect was most likely due to reduced vigilance and unsatisfactory pain relief. However, both effects significantly compromise lung function and the ability to cough.

12. **Stenseth et al, European Journal of Cardiothoracic Surgery. 1996; 10 (10): 859 – 65;** They conducted a study evaluating the effect of thoracic epidural analgesia on pulmonary function after coronary artery bypass surgery. Fifty-four male patients, under 65 years and with an ejection fraction of more than 0.5, were randomized into two groups: a control group receiving high-dose fentanyl anaesthesia and an epidural group receiving low-dose fentanyl anaesthesia + thoracic epidural analgesia. They concluded that thoracic epidural analgesia yields a slight, but significant, **improvement in pulmonary function**, most likely due to a more profound postoperative analgesia.

13. **H.Kehlet and K.Holte**; **British Journal of Anaesthesiology (2001) 87 (1): 62-72. .** Their article reviewed about the effect of postoperative analgesia on surgical outcome.

They concuded that the continuous epidural local anaesthetic or local anaesthetic–opioid mixtures have only been demonstrated to provide **a reduction in postoperative pulmonary morbidity** in major abdominal procedures.

14. Scott et al, British. Journal of Anaesthesiology: (1989) 62 (3) : 253-257

They conducted a randomized, double-blind study in twenty-two patients undergoing upper abdominal surgery who received extradural (T7–T8) 0.5% bupivacaine 9 ml followed by 25 mg h−1 with or without additional extradural morphine (bolus 4 mg plus 0.5 mg h−1), for 16 h after operation. There occured total alleviation of pain, and a stable level of sensory analgesia and **improvement in lung function.**

MATERIALS AND METHODS

A prospective randomised double blind study was done to evaluate the effect of epidural anaesthesia on pulmonary functions in patients undergoing upper abdominal surgery under general anaesthesia. The study was carried out in 82 adult patients at Govt Rajaji Hospital, Madurai. Patients in the age group of 20 – 60 years with BMI < 28 were selected for the study. Study was conducted after getting ethical committee approval.

Patients with chronic obstructive pulmonary disease, smokers, expected intraoperative blood loss > 500ml and skeletal deformities were excluded from the study. Only patients of ASA I and II physical status were chosen to avoid the influence of other medical illness. They were divided in to two groups with 42 patients in group GA and 40 patients in the other group EGA. For upper abdominal surgeries group GA were given general anaesthesia alone and group EGA were given both epidural and general anaesthesia.

PREANAESTHETIC EVALUATION

1. History
2. Clinical examination

3. Relevant investigations – Hemoglobin,Blood sugar, Renal function tests, Electrolytes, ECG, Chest x ray
4. Informed consent from patients

METHODS

All the patients were explained about the pulmonary function testing procedure completely and the same was demonstrated to all patients . Pulmonary function tests were performed in supine position preoperatively and 30 minutes after surgery (postoperatively). After attaching the nasal clip and placing the mouthpiece in the mouth with lips completely surrounding the mouthpiece, patient was instructed to take deep inspiration rapidly and exhale with maximal force without pause to get best results. The three parameters of forced indices FEV_1, FVC, PEFR were recorded. This manouvre was repeated three times and the best of three results was chosen.

EPIDURAL ANAESTHESIA

Epidural catheter was fixed in group EGA patients before induction. Tuohy needle (18 G) was introduced in the epidural space by the loss of resistance method at the level of T9 – T10 interspace. A catheter (20 G) was inserted through the needle and fixed with its tip at T6 – T7 interspace. Test dose of 3ml of 1.5% lignocaine with epinephrine 15mics was given to rule out intrathecal or intravascular spread. Intraoperatively

epidural anaesthesia was maintained with 0.25% bupivacaine every 40 minutes. Epidural analgesia was continued postoperatively for 24 hours after surgery with 0.125% bupivacaine.

GENERAL ANAESTHESIA

All patients were premedicated with inj. glycopyrrolate 0.2mg im and inj midazolam 0.05mg/kg im 45 minutes before surgery after performing pulmonary function test. All patients were induced with inj propofol 2mg/kg, inj fentanyl 2mics/kg, inj succinylcholine 2mg/kg iv and intubated. Inj fentanyl and inj atracurium were used for intraoperative maintenance in titrated doses. Patients were reversed with inj neostigmine 40mics/kg and inj glycopyrrolate 20mics/kg iv and extubated on table.

POSTOPERATIVE PERIOD

Pulmonary function tests were performed in the same manner as did in the preoperative period 30 mins after surgery. Three parameters of forced indices (FEV_1, FVC, PEFR) were recorded again and the best of three results were taken and compared.

ANALYSIS OF DATA

The observations were recorded for both the groups as shown in the master chart.

STATISTICAL TOOLS

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using Epidemiological Information Package (EPI 2010) developed by Centre for Disease Control, Atlanta.

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables and Yate's chi square test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

OBSERVATIONS AND RESULTS

The two groups were comparable in patient characteristics like age, sex, weight, and duration of surgery. The diagnosis and the procedure done are listed below.

A : CHARACTERISTICS OF CASES STUDIED :

TABLE – 1: AGE DISTRIBUTION

Age group	**G.A. group**		**EGA group**	
	No	**%**	**No**	**%**
Upto 20 years	1	2.4	1	2.5
21- 30 years	2	4.8	5	12.5
31-40 years	11	26.2	12	30
41-50 years	24	57.1	17	42.5
Above 50 years	4	9.5	5	12.5
Total	42	100	40	100
Range	20 - 55 years		19 - 55 years	
Mean	43.7 years		41.6 years	
SD	7.8 years		8.7 years	
'p'	0.284 **Not significant**			

The mean age of the General Anaesthesia Group was 43.7 $\pm$ 7.8 years and the Epidural General Anaesthesia Group was 41.6 $\pm$ 8.7 years. There was no statistically significant difference (p = 0.7447).

GRAPH-1:AGE DISTRIBUTION

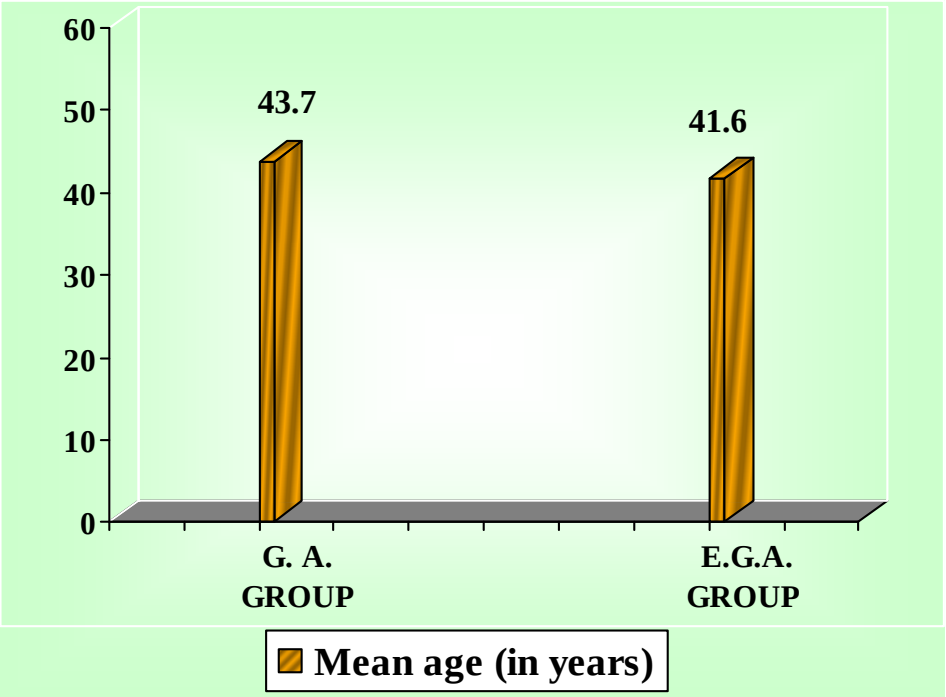


TABLE – 2: SEX DISTRIBUTION

Sex	G.A. group		EGA group	
	No	**%**	**No**	**%**
Male	24	57.1	20	50
Female	18	42.9	20	59
Total	42	100	42	100
'p'	0.6695 **Not significant**			

57.1% of the G.A. group and 50% of the E.G.A. group were males. There was no significant difference in the sex composition of the two groups. ('p' > 0.05)

GRAPH-2:SEX DISTRIBUTION

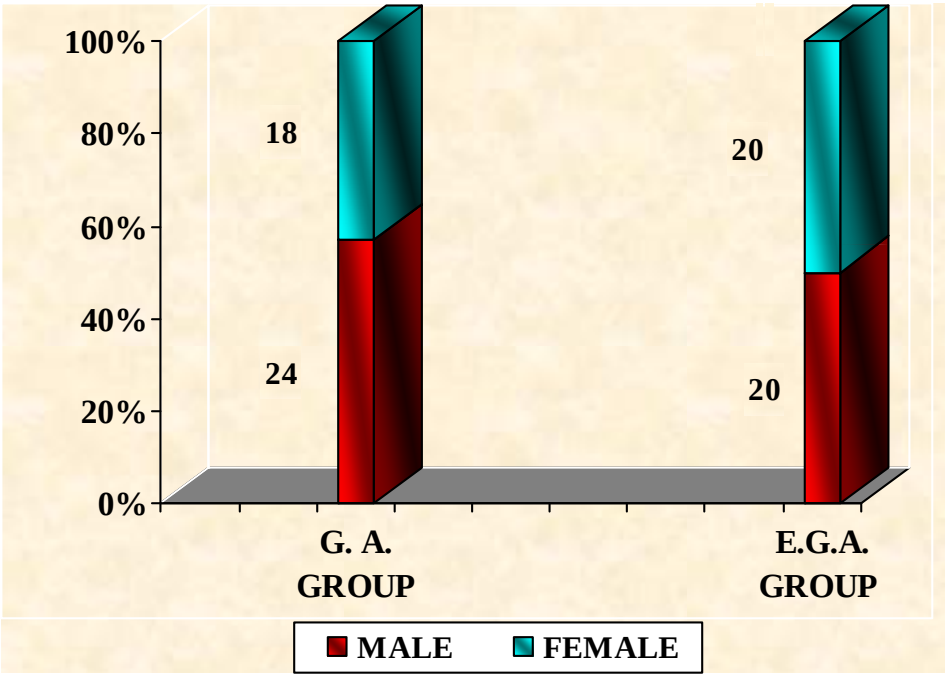


TABLE 3: DIAGNOSIS

Diagnosis	G.A. group		EGA group	
	No	%	No	%
Ca. Stomach	**12**	**28.6**	**10**	**25**
Cholelithiasis	**10**	**23.8**	**13**	**32.5**
Goo	**12**	**28.6**	**9**	**22.5**
Others	**8**	**19.0**	**8**	**20**
Ca. Easophagus	1	2.4	-	-
CBD stone	1	2.4	-	-
Adhesive obstruction	1	2.4	-	-
Hep. Flex growth	1	2.4	-	-
Ileocecal TB stricture	2	4.8	1	2.5
Koch abdomen	1	2.4	1	2.5
Linear abscess	-	-	3	7.5
Splenic flexure growth	-	-	1	2.5
Sigmoid colon growth	-	-	1	2.5
Trans colon growth	1	2.4	1	2.5
Total	**42**	**100**	**40**	**100**

GRAPH-3:DIAGNOSIS

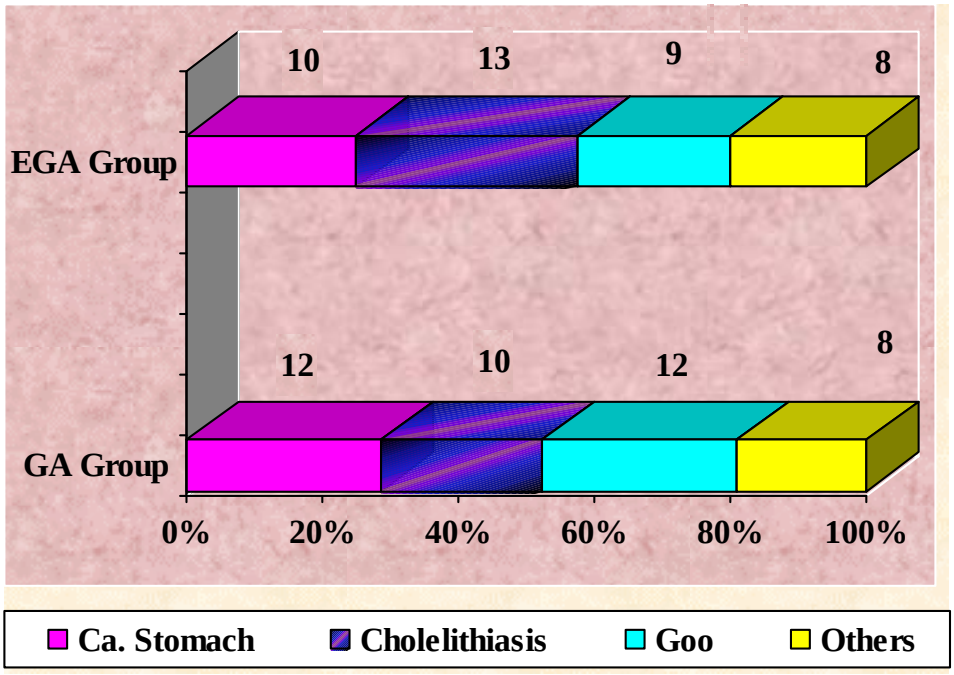


TABLE 4 : PROCEDURE

Procedure	G.A. group		EGA group	
	No	%	No	%
Cholecystectomy	**11**	**26.2**	**13**	**32.5**
Laparotomy	**16**	**38.1**	**13**	**32.5**
TVGJ	**12**	**28.6**	**9**	**22.5**
Others	**3**	**7.1**	**5**	**12.5**
Adhesiolysis	1	2.4	-	-
Diagnostic laparotomy	1	2.4	1	2.5
Liver abscess drainage	-	-	3	7.5
Resection and anastomosis	-	-	1	2.5
Trans Hiatal Oesophagectomy	1	2.4	-	-
Total	**42**	**100**	**40**	**100**

GRAPH-4:PROCEDURE

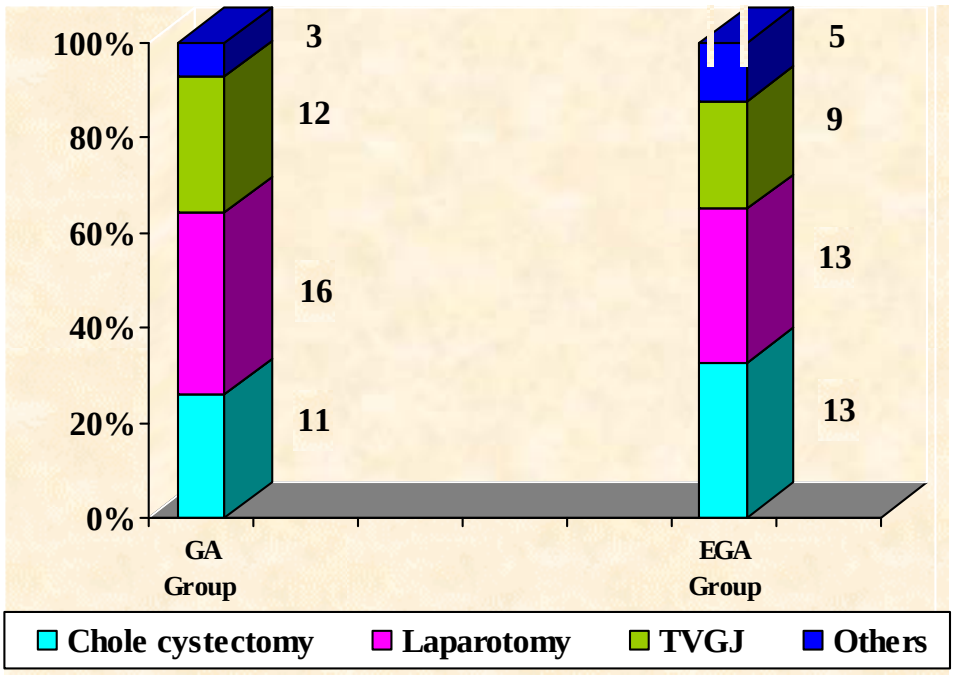
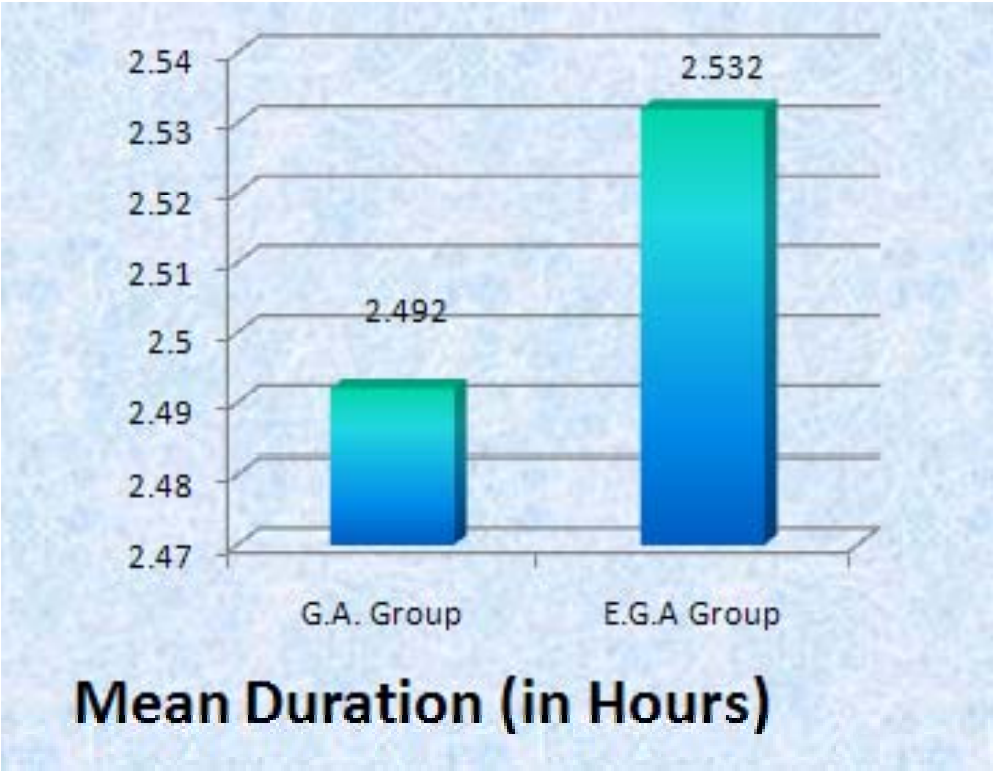


TABLE 5: DURATION OF SURGERY (in Hours)

Duration in Hours	G.A. group	EGA group
Range	2.1 – 3.5	1.9 – 3.2
Mean	2.49	2.53
SD	0.27	0.26
'p'	0.5 **Not significant**	

GRAPH-5:DURATION OF SURGERY



B: CHANGES IN PRE OPERATIVE AND POST OPERATIVE PULMONARY FUNCTION TESTS

TABLE – 5: CHANGES IN FEV_1

(In percentage of predicted values)

FEV_1 Value	FEV_1 values (in %)						'p'
	GA group			EGA group			
	Range	Mean	SD	Range	Mean	SD	
Pre operative	62 - 78	71.0	4.3	60 - 80	69.6	4.8	0.1167 Not significant
Post operative	17 - 46	35.4	6.3	34 - 63	44.8	6.8	**0.0001 Significant**
Difference	16 - 48	35.6	6.6	14 - 32	24.8	4.2	**0.0001 Significant**

Percentage of decrease in FEV_1 values were 24.8%$\pm$4.2% in EGA group whereas they were 35.6%$\pm$6.6% in the GA group. This is statistically significant. ('P'=0.0001)

GRAPH-6:CHANGES IN FEV_1

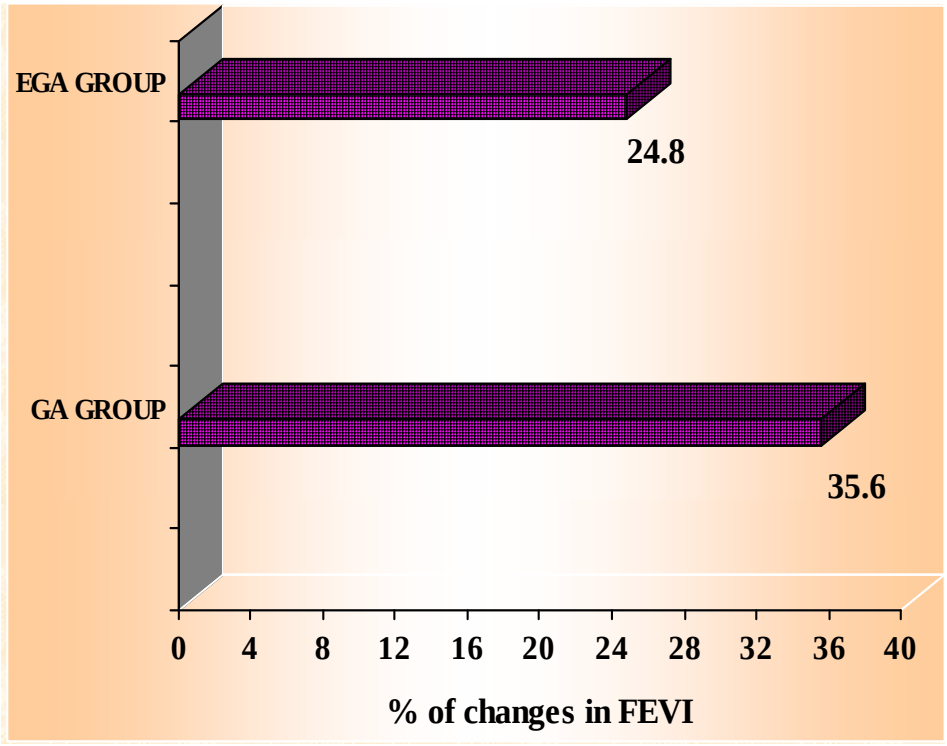


TABLE – 6: CHANGES IN FVC

FVC	FVC values (in %)						'p'
	GA group			EGA group			
	Range	Mean	SD	Range	Mean	SD	
Pre operative	60 - 79	70.4	5.7	60 - 80	68.4	5.5	0.0948 Not significant
Post operative	17 - 47	34.1	6.1	30 - 56	43.3	6.1	**0.0001 Significant**
Difference	26 - 54	36.3	5.7	12 - 35	25.1	4.6	**0.0001 Significant**

FVC values decreased by 36.3%±5.7% in the GA group of patients. But the decrease was only 25.2%± 4.6% in the EGA group which was statistically significant.

GRAPH-7:CHANGES IN FVC

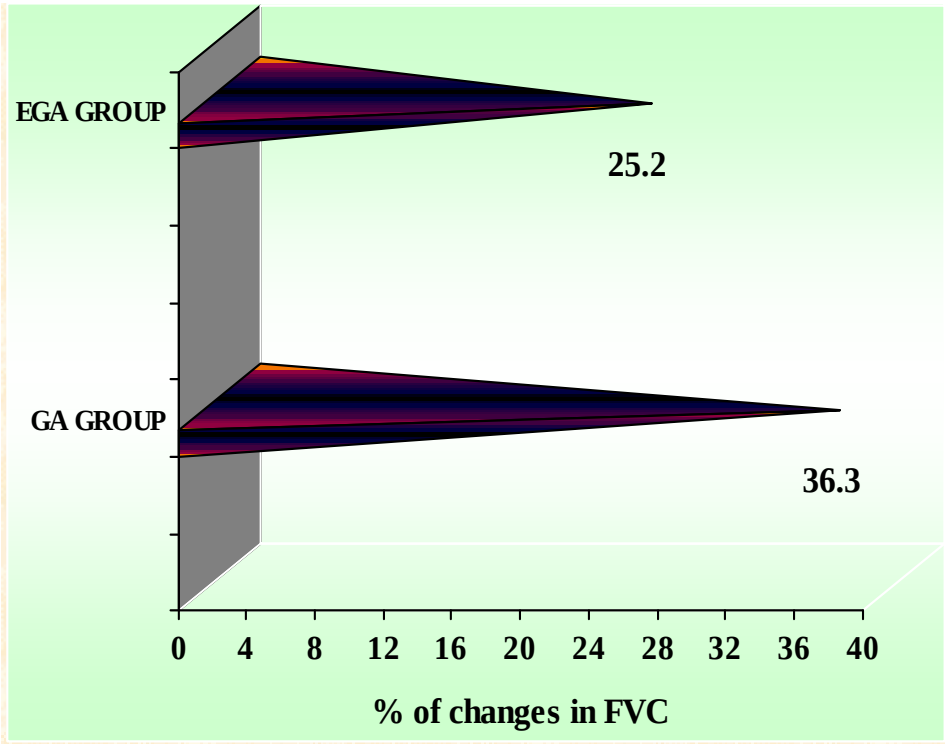
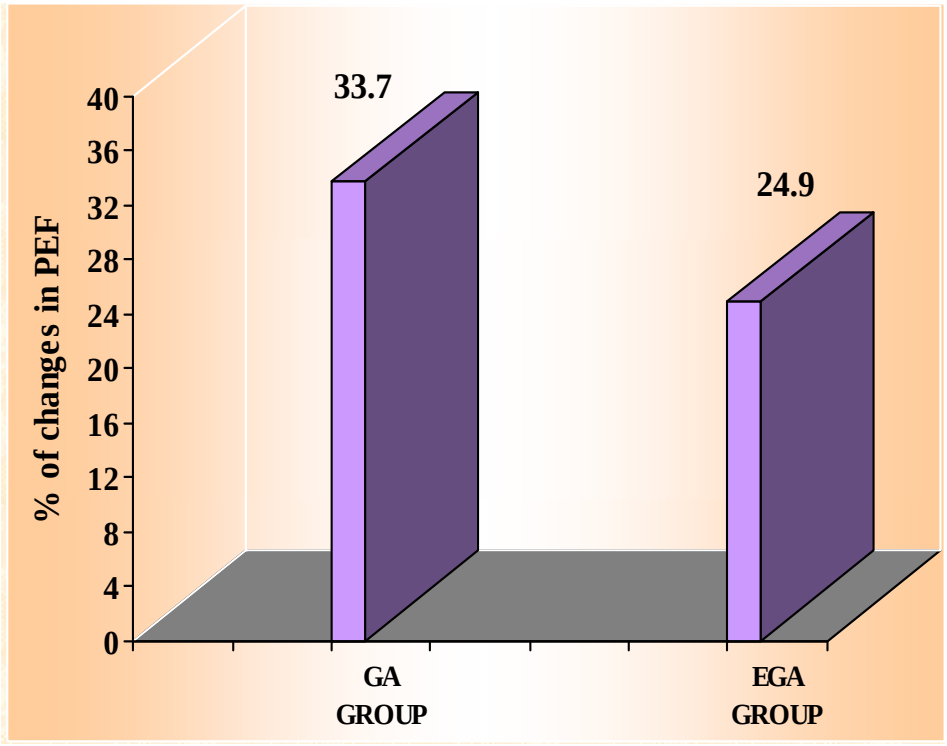


TABLE – 7: CHANGES IN PEFR

PEFR	PEFR values (in %)						'p'
	GA group			EGA group			
	Range	Mean	SD	Range	Mean	SD	
Pre operative	60 - 80	70.4	6.0	60 - 80	69.0	6.0	0.2173 Not significant
Post operative	11 - 50	36.7	9.0	20 - 57	44.1	8.3	**0.0001 Significant**
Difference	22 - 49	33.7	6.2	17 - 42	24.9	5.4	**0.0001 Significant**

The decrease in PEFR values in the GA group was 33.7% $\pm$ 6.2%. This is significantly higher than the decrease in the EGA group (24.9% $\pm$ 5.4%)

GRAPH-8:CHANGES IN PEFR



DISCUSSION

Postoperative respiratory dysfunction is universally observed after abdominal and thoracic surgery. Abnormalities that contributes to reduced lung volume and hypoxaemia in postoperative period include impaired central ventilatory control, abnormal pulmonary mechanics due to limited abdominal, intercostals, and diaphragmatic muscle contraction and changes in pulmonary circulation and gas exchange. These abnormalities are due not only to sequele of operation itself, such as tissue injury or pain, but also to residual effects of anaesthesia and analgesia.

It is necessary to alleviate pain and improve the respiratory muscle tone as early as possible to prevent postoperative pulmonary complications and promote early recovery. It is a well known fact that epidural technique is simple to perform, safer in skilled hands, and less time consuming. By this epidural analgesia, postoperative problems of major abdominal surgeries are overcome.

In this study, forced indices are used to compare the pulmonary function tests in postoperative period as they maximally depend on respiratory muscular effort and are affected the most in this period. Other

pulmonary function test parameters are least affected in the immediate postoperative period.

The two groups were comparable with respect to age, sex, weight and duration of surgery.

Since in the immediate postoperative period it will be cumbersome for the patient to do the spirometry test in sitting position with epidural anaesthesia, supine position was chosen in this study for both preoperative as well as postoperative period. As the procedure was done in supine posture, the initial preoperative pulmonary function tests results were reduced than the expected 80% of the predicted value. Hence the study was standardized in such a way that all patients in the study group underwent the pulmonary function tests in supine posture both in preoperative and postoperative period.

FORCED EXPIRATIORY VOLUME IN 1 SECOND:

The preoperative FEV_1 in both groups (GA and EGA) were comparable. In the postoperative period, patients in EGA group performed the pulmonary function tests well compared with GA group. The reduction in postoperative FEV_1 from the preoperative value is only $24.8 \pm 4.2\%$ in group EGA compared with group GA where the reduction was 35.6+6.6% and which was statistically significant.

This outcome is comparable with the study conducted by H.Hendolin and his colleagues, which was published in Acta Anaesthesiologica Scandinavica which concluded TEA significantly prevented the postoperative deterioration of respiratory function as compared with general anaesthesia.

FORCED VITAL CAPACITY

The preoperative value of FVC both in group GA patients and in group EGA patients were comparable. The postoperative value of FVC was reduced by 36.3%$\pm$5.7% from the preoperative value in the GA group whereas in group EGA the reduction of FVC from the preoperative value was only 25.1%$\pm$4.6% which again goes by the study conducted by H.Hendolin et al and Guay Joahne et al which concluded that TEA improves the forced vital capacity (FVC), (P = 0.001) at 24 h.

PEAK EXPIRATORY FLOW RATE

The preoperative values of PEFR were comparable in both GA and EGA group patients. There occurred 33.7%$\pm$6.2% reduction of PEFR in the postoperative period in group GA patients. Whereas only 24.9%$\pm$5.4% reduction of PEFR occurred in group EGA patients.

Hence it is clear from the study that overall pulmonary function improve even in the immediate postoperative period with epidural anaesthesia

compared with general anaesthesia only. This study correlates with the study conducted by Scott et al and Daniel M Popping et al which concludes the overall improvement in lung function with epidural anaesthesia in the postoperative period.

SUMMARY

82 patients of ASA (I &II) undergoing upper abdominal surgeries were randomly assigned in two groups, Group GA (General anaesthesia only) and Group EGA (Epidural and General anaesthesia)

Patients of both the groups were explained and demonstrated about the pulmonary function testing procedure. Pulmonary function test was done using timed spirogram and the values of forced indices (FEV_1,FVC, PEFR) were recorded before surgery and 30 minutes after surgery.

The observations were:

1. Reduction of FEV_1 by 29% - 41% in patients of GA group whereas only 20% - 28% reduction in EGA group.
2. Reduction of FVC by 31% - 41% in patients of GA group whereas only 21% - 29% reduction in EGA group.
3. Patients of GA group showed reduction of PEFR by 28% - 40% whereas in EGA group PEFR reduced only by 20% - 30%

Hence it can be concluded that epidural analgesia reduces the impact of general anaesthesia and pain on pulmonary function in the postoperative period.

CONCLUSION

Epidural anaesthesia can be provided safely in appropriate patients undergoing major abdominal surgery. It offers a number of proven benefits as a result of pain relief, improving the respiratory muscle tone and hence pulmonary function. This study concluded that pulmonary functions are significantly improved by epidural anaesthesia, thus emphasizing epidural anaesthesia can significantly decrease pulmonary morbidity.

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PROFORMA

EVALUATION OF EFFECT OF EPIDURAL ANAESTHESIA ON PULMONARY FUNCTIONS IN PATIENTS UNDERGOING UPPER ABDOMINAL SURGERY UNDER GENERAL ANAESTHESIA

NAME: AGE: SEX: WEIGHT:

DIAGNOSIS:

PROCEDURE:

DURATION OF SURGERY:

ANAESTHESIA : GA / EPIDURAL – GA

PREOPERATIVE PULMONARY FUNCTION TEST

FORCED INDICES	FIRST RECORD	SECOND RECORD	THIRD RECORD
FEV1			
FVC			
PEFR			

POSTOPERATIVE PUMONARY FUNCTION TEST

FORCED INDICES	FIRST RECORD	SECOND RECORD	THIRD RECORD
FEV1			
FVC			
PEFR			

MASTER CHART

GROUP GA

S NO	GROUP	NAME	AGE	SEX	IP NO	Diagnosis	Procedure	Duration (in hrs)	PRE Operative			POST Operative		
									FEV1	FVC	PEF	FEV1	FVC	PEF
1	GA	MAHALINGAM	54	M	3752	CA OESOPHAGUS	TRANS HIATAL OESOPHAGECTOMY	3.5	73	64	72	40	32	37
2	GA	PADMAVATHY	45	F	5912	GOO	TVGJ	2.3	70	60	63	35	30	34
3	GA	PANDIAMMAL	42	F	5668	GOO	TVGJ	2.5	71	79	67	30	25	20
4	GA	SELVAM	38	M	2656	CHOLELITHIASIS	CHOLECYSTECTOMY	2.7	70	60	78	36	32	40
5	GA	KARUPPAN	50	M	6430	CA STOMACH	LAPARATOMY	2.4	66	64	63	38	35	30
6	GA	SUNDARI	55	F	6811	CA STOMACH	LAPARATOMY	2.3	78	72	62	30	28	28
7	GA	SARAVANA KUMAR	32	M	9455	KOCH ABDOMEN	DIAGNOSTIC LAPARATOMY	2.5	76	76	64	31	29	17
8	GA	ANBURAJ	55	M	7513	CA STOMACH	LAPARATOMY	2.6	78	70	64	35	29	24
9	GA	DEVRAJ	45	M	6215	GOO	TVGJ	2.8	62	61	60	17	17	11
10	GA	VAIRAMANI	46	F	6349	CHOLELITHIASIS	CHOLECYSTECTOMY	2.4	76	71	62	46	40	35
11	GA	SUBBAIAH	50	M	O380	CA STOMACH	LAPARATOMY	2.8	76	72	66	30	34	40
12	GA	MOHAN	48	M	1095	GOO	TVGJ	2.1	70	72	78	46	38	50
13	GA	PAPPA	50	F	2981	CA STOMACH	LAPARATOMY	2.4	68	72	64	28	34	28
14	GA	SHANMUGAPATHY	45	M	2013	CA STOMACH	LAPARATOMY	2.5	76	70	68	40	44	46
15	GA	AYYAKANNU	40	M	4526	GOO	TVGJ	2.3	77	78	70	46	36	40
16	GA	RAMAN	40	F	6349	GOO	TVGJ	2.2	70	76	79	34	30	46
17	GA	RUKMANI	50	F	5188	CHOLELITHIASIS	CHOLECYSTECTOMY	2.9	66	62	70	28	30	28
18	GA	ANBURAJ	45	M	7517	CA STOMACH	LAPARATOMY	2.4	72	76	76	40	40	45
19	GA	ACHIMUTHU	38	M	8409	CHOLELITHIASIS	CHOLECYSTECTOMY	2.3	70	78	78	46	47	50
20	GA	KAMARAJ	46	M	5108	GOO	TVGJ	2.5	78	74	70	36	40	40

GROUP GA

SNO	GROUP	NAME	AGE	SEX	IP NO	Diagnosis	Procedure	Duration (in hrs)	PRE Operative			POST Operative		
									FEV1	FVC	PEF	FEV1	FVC	PEF
21	GA	FATHIMA	40	F	4460	CHOLELITHIASIS	CHOLECYSTECTOMY	2.5	70	78	78	35	40	45
22	GA	RAVI	31	M	12993	ILEOCECAL TB-STRICTURE	LAPARATOMY	2.4	71	76	78	46	36	40
23	GA	PADMAVATHI	50	F	13180	CHOLELITHIASIS	CHOLECYSTECTOMY	2.5	68	66	66	30	28	38
24	GA	VEERALAKSMI	20	F	15107	CHOLELITHIASIS	CHOLECYSTECTOMY	2.7	62	78	80	46	47	48
25	GA	MARUDHU	45	M	15009	GOO	TVGJ	2.3	66	64	60	24	31	35
26	GA	RAJATHI	48	F	15987	GOO	TVGJ	2.3	70	66	68	32	32	36
27	GA	PANDI	38	M	16103	GOO	TVGJ	2.5	76	70	76	30	28	34
28	GA	RAAKKU	50	F	17480	CA STOMACH	LAPARATOMY	2.2	64	60	66	26	25	30
29	GA	PANDIAMMAL	46	F	17704	CA STOMACH	LAPARATOMY	2.8	70	74	68	34	32	35
30	GA	SUNDARAM	40	M	17632	GOO	TVGJ	2.4	72	76	77	38	40	44
31	GA	JAYARAM	45	M	36767	CHOLELITHIASIS	CHOLECYSTECTOMY	2.6	72	70	78	38	38	40
32	GA	DANABAKIAM	50	F	38843	CBD STONE	CHOLECYSTECTOMY	2.1	70	68	72	36	34	30
33	GA	BAGAVATY	26	F	40057	ADHESIVE OBSRUCTION	ADHESIOLYSIS	2.4	78	77	78	40	40	45
34	GA	MAGESWARI	30	F	37455	CA STOMACH	LAPARATOMY	2.9	68	70	70	34	30	40
35	GA	JEGANADHAN	49	M	35858	CA STOMACH	LAPARATOMY	2.3	66	68	70	38	35	30
36	GA	PALANI	55	M	36348	ILEOCECAL TB - STRICTURE	LAPARATOMY	2.7	68	66	70	35	32	32
37	GA	KURUVAN	43	M	39945	CHOLELITHIASIS	CHOLECYSTECTOMY	2.3	74	76	70	36	36	40
38	GA	CHELLAMAL	45	F	36594	HEP FLEXURE GROWTH	LAPARATOMY	2.4	67	70	68	35	36	40
39	GA	SIVALINGAM	37	M	39998	CHOLELITHIASIS	CHOLECYSTECTOMY	2.1	74	70	80	36	38	50
40	GA	CHINAPPAN	50	M	41199	GOO	TVGJ	3	68	64	65	33	28	30
41	GA	PANDIAMAL	40	F	35903	CA STOMACH	LAPARATOMY	2.3	73	75	72	38	40	44
42	GA	RAMASAMY	43	M	37568	TRANS COLON GROWTH	LAPAROTOMY	2.6	70	68	73	34	38	45

GROUP EGA

S NO	GROUP	NAME	AGE	SEX	IP NO	Diagnosis	Procedure	Duration (in hrs)	PRE Operative			POST Operative		
									FEV1	FVC	PEF	FEV1	FVC	PEF
43	EGA	PARAMESWARI	37	F	5649	CHOLELITHIASIS	CHOLECYSTECTOMY	2.2	75	77	61	45	42	20
44	EGA	PONNUTHAI	55	F	5723	GOO	TVGJ	2.7	72	61	62	43	38	20
45	EGA	RAJESWARI	40	F	54278	CHOLELITHIASIS	CHOLECYSTECTOMY	2.5	73	66	63	46	54	33
46	EGA	PRABAKARAN	35	M	92184	LIVER ABSCESS	DRAINAGE	2.1	72	61	60	50	42	43
47	EGA	MADHAVI	37	F	5466	CHOLELITHIASIS	CHOLECYSTECTOMY	2.6	77	77	62	63	54	41
48	EGA	SUGANTHI	28	F	93039	KOCH ABDOMEN	DIAGNOSTIC LAPARATOMY	2.7	67	62	65	42	39	40
49	EGA	MANIMEGALAI	29	F	15818	CHOLELITHIASIS	CHOLECYSTECTOMY	2.7	78	76	80	56	50	57
50	EGA	PAPPATHI	38	F	12309	CA STOMACH	LAPARATOMY	2.4	76	74	77	48	48	44
51	EGA	KALAIAMMAL	54	F	17304	CA STOMACH	LAPARATOMY	2.9	68	66	78	36	40	48
52	EGA	KARUPUDHEVAR	52	M	18690	GOO	TVGJ	2.5	64	66	60	40	43	32
53	EGA	JABEELA	45	F	17970	CHOLELITHIASIS	CHOLECYSTECTOMY	2.7	66	68	70	48	46	48
54	EGA	MALAICHAMY	40	M	21296	GOO	TVGJ	2.7	74	78	77	50	52	50
55	EGA	CHIDAMBARAM	42	M	20276	TRANS COLON GROWTH	LAPAROTOMY	3.2	70	64	78	52	36	49
56	EGA	THANGARAJ	51	M	27378	CA STOMACH	LAPARATOMY	2.9	66	65	70	34	38	40
57	EGA	SEENIAMMAL	50	F	25051	ILEOCECAL TB - STRICTURE	LAPARATOMY	2.4	64	66	66	40	36	38
58	EGA	MANIMALA	28	F	31396	CHOLELITHIASIS	CHOLECYSTECTOMY	2.6	78	77	76	52	48	56
59	EGA	MURUGESAN	35	M	31376	GOO	TVGJ	2.5	78	74	75	50	50	49
60	EGA	PARGAVI	43	F	31960	GOO	TVGJ	2.6	68	66	68	48	38	42
61	EGA	ARULAPPAN	40	M	32006	CHOLELITHIASIS	CHOLECYSTECTOMY	2.3	70	74	77	48	50	54
62	EGA	GANESAN	46	M	32405	CA STOMACH	LAPARATOMY	2.4	68	66	70	40	40	48

GROUP EGA

S NO	GROUP	NAME	AGE	SEX	IP NO	Diagnosis	Procedure	Duration (in hrs)	PRE Operative			POST Operative		
									FEV1	FVC	PEF	FEV1	FVC	PEF
63	EGA	SHANTHI	45	F	41545	CHOLELITHIASIS	CHOLECYSTECTOMY	2.3	68	70	66	44	50	42
64	EGA	UMASANKARI	32	F	43143	CHOLELITHIASIS	CHOLECYSTECTOMY	2.6	73	70	72	50	48	52
65	EGA	TAMIL SELVAM	19	M	42742	CHOLELITHIASIS	CHOLECYSTECTOMY	2.5	80	80	78	60	56	56
66	EGA	PERUMAL	37	M	43236	CHOLELITHIASIS	CHOLECYSTECTOMY	2.1	68	63	66	36	30	35
67	EGA	MOSES	50	M	43310	CA STOMACH	LAPARATOMY	2.9	64	68	64	35	36	43
68	EGA	GUNASEKARAN	50	M	44384	GOO	TVGJ	2.8	68	60	67	42	43	42
69	EGA	PERUMAL	50	M	44397	CA STOMACH	LAPARATOMY	2.6	65	68	66	40	43	45
70	EGA	VIJAYA	52	F	46587	CHOLELITHIASIS	CHOLECYSTECTOMY	2.5	60	61	63	36	34	40
71	EGA	RAMUTHAI	40	F	45761	SPLENIC FLEXURE GROWTH	LAPARATOMY	2.8	70	68	72	45	42	48
72	EGA	SUSAN	48	M	47684	CA STOMACH	LAPARATOMY	2.6	66	65	67	38	40	43
73	EGA	DEEPA	25	F	45740	CHOLELITHIASIS	CHOLECYSTECTOMY	2.4	76	74	77	50	46	55
74	EGA	VAIRAMANI	29	M	46757	LIVER ABSCESS	DRAINAGE	2.0	70	75	77	50	49	51
75	EGA	BALALAKSMI	47	F	44590	SIGMOID COLON GROWTH	RESECTION AND ANASTAMOSIS	2.7	66	65	62	36	40	39
76	EGA	RAMAKRISHNAN	44	M	36740	GOO	TVGJ	2.5	68	65	70	45	42	48
77	EGA	IRULAYEE	45	F	46630	CA STOMACH	LAPARATOMY	2.3	67	70	66	38	36	40
78	EGA	MARIMUTHU	48	M	45825	LIVER ABSCESS	DRAINAGE	1.9	63	66	69	39	41	45
79	EGA	MUTHAIYA	46	M	50617	GOO	TVGJ	2.8	68	65	66	40	41	45
80	EGA	CHINNAPONNU	45	F	50823	CA STOMACH	LAPARATOMY	2.6	65	69	67	47	50	50
81	EGA	DURAIPANDI	50	M	48259	GOO	TVGJ	2.3	66	60	63	44	40	45
82	EGA	PANDI	36	M	48445	CA STOMACH	LAPARATOMY	2.5	70	71	68	46	40	49

EVALUATION OF EFFECTS OF EPIDURAL ANAESTHESIA ON PULMONARY FUNCTION IN PATIENTS UNDERGOING UPPER ABDOMINAL SURGERY UNDER GENERAL ANAESTHESIA.

ABSTRACT

Background

This is a randomized, prospective study of evaluation of effects of epidural anaesthesia on pulmonary function in patients undergoing upper abdominal surgery under general anaesthesia.

Methods

82 patients of ASA (I &II) undergoing upper abdominal surgeries were randomly assigned in two groups, Group GA (General anaesthesia only/42 patients) and Group EGA (Epidural and General anaesthesia/40 patients). Patients of both the groups were explained and demonstrated about the pulmonary function testing procedure. Pulmonary function test was done using timed spirogram and the values of forced indices (FEV_1, FVC,PEFR) were recorded before surgery and 30 minutes after surgery.

Observations

Pulmonary function indices significantly improved in the group EGA after surgery. Reduction of FEV_1 by 29% - 41% in patients of GA group whereas only 20% - 28% reduction in EGA group ($p<0.05$). Reduction of FVC by 31% - 41% in patients of GA group whereas only 21% - 29% reduction in EGA group ($p<0.05$). Patients of GA group showed reduction of PEFR by 28% - 40% whereas in EGA group PEFR reduced only by 20% - 30% ($p<0.05$).

Conclusion

Epidural analgesia significantly reduces the impact of general anaesthesia and pain on pulmonary function in the postoperative period.

Key words

Pulmonary function test, Epidural analgesia, general anaesthesia, timed spirogram